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Eating disorders associated with polycystic ovary syndrome (PCOS), a literature review

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ABSTRACT

Polycystic ovary syndrome (PCOS) is a common enigmatic, heterogenous endocrine disorder with multisystem consequences affecting women at reproductive age. Most associated dysfunctions in PCOS patients include ovarian dysmorphology, hormonal imbalances, metabolic disturbances, and neuropsychological impairments. Emerging studies have shown a relationship between women with PCOS and eating disorders (ED). ED is described as a psychological disorder characterized by extreme and constant disruptions in eating behaviors that can negatively impact one's mental health and status. Types of EDs include binge eating disorder (BED), bulimia nervosa (BN), anorexia nervosa (AN), other specified feeding or eating disorders (OSFED), and unspecified feeding and eating disorders (UFED). Understanding the clinical relevance of these associations and adding correct assessments of ED as a key determinant of eating behavior may contribute to the successful treatment of women with PCOS. Thus, this review aims to outline a more profound connection between the involvement of multiple eating disorders and related disordered eating in women with PCOS; it also establishes a wider overview regarding the prevalence of comorbidities in eating disorders. Furthermore, the majority of researchers advised that screening for eating disorders, PCOS phenotyping, and neuroimaging modalities can be utilized to provide a better health outcome in PCOS patients. PubMed and Web of Science databases were searched for reviews on this issue.

Keywords: Polycystic ovary syndrome, PCOS, eating disorders, bulimia nervosa, anorexia nervosa

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a heterogenous multisystem disorder mostly affecting women of reproductivewith an increasing prevalence recently estimated to be 10%-13%.[1-3] PCOS is characterized by a diversity of endocrine-metabolic, psychological, and reproductive manifestations, examples of each including hyperandrogenism-hyperinsulinism, poor self-esteem, and polycystic ovarianmorphological abnormalitiesrespectively. In addition to these factors, clinical features like hirsutism, acne appearance, elevated body mass index (BMI), and obesity have contributed to adverse eating psychological disorders that have become progressively apparent in patients with PCOS.[4]

Eating disorders(ED) are a broad spectrum of complex psychological disorders with a strong female predominance that is characterized by abnormal eating behaviors that may or may not be accompanied by compensatory behaviors.[4,5] Various types of eating disorders include bulimia nervosa (BN), anorexia nervosa (AN), binge eating disorder (BED),[5-7] other specified feeding or eating disorders (OSFED),[7,8] and unspecified feeding and eating disorder (UFED).[8,9] BED is a condition marked by overeating without subsequent purging.[9] AN is characterized by severely limiting energy intake, which leads to low body weight, along with strong anxiety about gaining weight and distorted perceptions of body shape.[10,11] BN occurs recurrently by having binge eating episodes with loss of control when these episodes happen andis accompanied by improper coping mechanisms.[7,11,12] Globally, the estimated prevalence of any ED is around 1%.[5] These disorders are most likely the result of complex interactions between biological, social, cultural, as well as individual factors.[13,14] Food restriction, binge eating, purging, the use of laxatives, diet medication, and excessive exercise are all behaviors that fall under the category of eating disorders.[4] The impact of an ED and its symptoms on all aspects of a person's life has been widely documented, with studies finding decreased interpersonal functioning as well as mental and physical concerns that are associated with higher morbidity and healthcare expenses.[15-18] As a result,

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eating disorders exacerbate obesity and metabolic syndrome, creating a perpetual vicious cycle[19].BED is the most common ED associated with women suffering from PCOS.[5] Overall, this present review adopts an integrative theoretical conception that aspires to shed light on the co-existence of different types of ED (BED, BN, EED, NES, AN) and (PCOS). PubMed and Web of Science databases were searched for reviews on this issue.

PATHOPHYSIOLOGY

PCOS

PCOS is characterized by irregular ovulation, increased androgens levels, abnormal GnRH pulsation, and insulin insensitivity.[20] PCOS is characterized by a reciprocal relationship between hyperandrogenism and insulin resistance, with higher levels of insulin leading to the development of hyperandrogenism and vice versa (Figure 1). Furthermore, dysfunction in the hypothalamus-pituitary-adrenal (HPA) axis results in elevated levels of androgens and insulin resistance. These factors all contribute to the development of PCOS.[20]

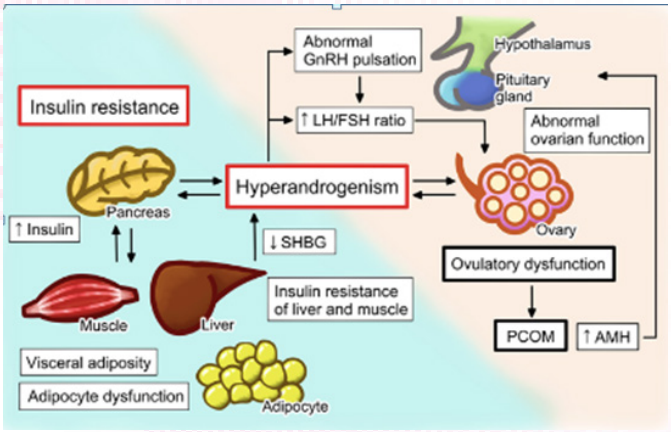

Figure 1. Effects of Insulin resistance and hyper and rogenism[20]

In addition to these associated contributors, it has been observed that there is a strong familial pattern in patients with this condition.[21] PCOS mothers show high androgenic hormone levels in the uterus resulting from raised androgen levels and impaired placental functioning. This in turn leads to the fetus' ovaries secreting increased levels of androgens in response to the intrauterine atmosphere of mothers with PCOS.[21] Research has indicated that prenatal androgen exposure can upset the intestinal microbial balance, potentially leading to PCOS later on.[20] Further, studies have suggested that PCOS and elevated concentrations of androgen in the uterus may increase the odds of their offspring suffering from neuropsychiatric disorders, for example, autism, ADHD, and Tourette Syndrome.[21]

Studies with pregnant mice showed that supplying the uterus with Antimüllerian hormone (AMH) caused offspring to exhibit signs of PCOS, such as hyperandrogenism and enlarged LH pulses, leading to reduced ovulation in female offspring (Figure 2). Those with anovulatory PCOS have more AMH than those with ovulatory PCOS.

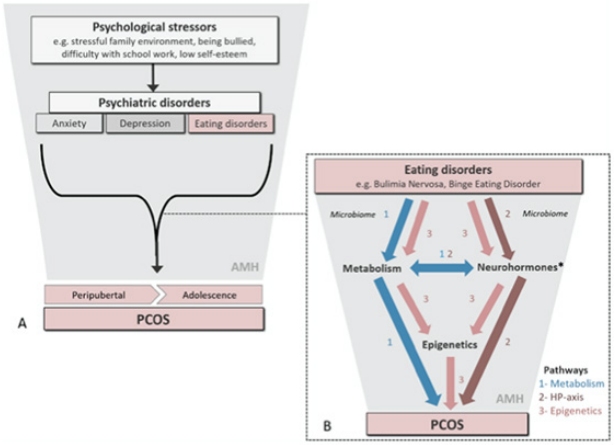

Figure 2. Effects of psychological stressors on developing of eating disorders in PCOS patients[14]

Psychosomatic Aspects in PCOS

Manifestations of psychological issues among patients with PCOS may be attributed to the connection between the mind and body, which can occur through three main pathways. These includedysregulation of neurotransmitters which is the main attribute of PCOS, instability in the neuroendocrine axis, and disruptions in the brain-gut axis.[4]

Neurotransmitter dysregulation: Neurotransmitters appear to be involved in sustaining mental equilibrium and some psychiatric issues. Major depression is attributed to an impairment of the monoamine systems, notably serotonin (5-HT), noradrenaline (NE), and dopamine (DA). Other than depression, imbalances in glutamate, gamma aminobutyric acid (GABA), and acetylcholine levels can lead to anxiety-related disorders. It also appears that those with PCOS and depression may have altered morphine levels. Generally, neurotransmitter imbalances can lead to reduced self-worth, anxiety, emotional fluctuations, irritability, and depression. Low concentrations of these mediators can cause irregular LH release in PCOS patients, potentially resulting in a neuroendocrine disorder.[4]

Neuroendocrine axis instability: The CNS's androgen receptors, insulin receptors, GABA transmissions, and internal opioid signaling likely contribute to PCOS's development, suggesting a neuroendocrine origin.[22] It is thought that an overactive HPA axis is the main source of PCOS symptoms. This heightened exercise can cause androgen hormone levels to rise, GnRH secretion to increase, and cortisol turnover to accelerate. Women with PCOS are prone to heightened depression, anxiety, and appetite due to the psychological repercussions and HPA axis activation, which is necessary to release FSH and LH gonadotropins.[4]

Additionally, the stimulation of GnRH neurons with AMH increases their rate of discharge, which leads to greater LH secretion and reduced FSH output from the pituitary. High gonadotropin levels, mainly LH, can lead to an excessive amount of androgens,[23] which could worsen depressive symptoms,[14] signifying that raised androgen levels may be the cause of PCOS-related psychiatric issues.[24] All in all, there seems to be a clear connection between the intensity of depression symptoms and androgens.[13,24]

Studies have demonstrated the connection between cortisol and depression,[13] as well as anxiety being associated with low serotonin and high cortisol levels.[14] Research suggests that individuals with AN[11] and BED have an above-average amount of cortisol, regardless of depression.[14] In contrast, cortisol levels in individuals with bulimia nervosa (BN) were regular.[13]

As a result of PCOS patients' hyperinsulinemia, reactive hypoglycemia can be caused, which then leads to the secretion of hormones such as adrenaline and cortisol that may bring about heart palpitations, tremors, anxiety, and irritability, ultimately causing disturbed sleep.[10] To understand how these connections affect the dietary habits of individuals with PCOS further investigation is needed.[25]

Disruptions in the brain-gut axis: Gut-brain axis exploration has unveiled essential insights with impacts on gastroenterology and neurology, especially psychiatry. Any disruption in the digestive system's operation can affect the brain's hormonal and automatic responses. The interaction between the digestive system and the brain is known as the gut–brain axis (Figure 3). The intricate combination of gut-derived-peptides(GDPs), CNS, and the gut-brain axis is critical for PCOS growth, maintenance, aberrant pathways, and psychological components.[4] These linked GDPs play an important part in the source and development of ED.[14]

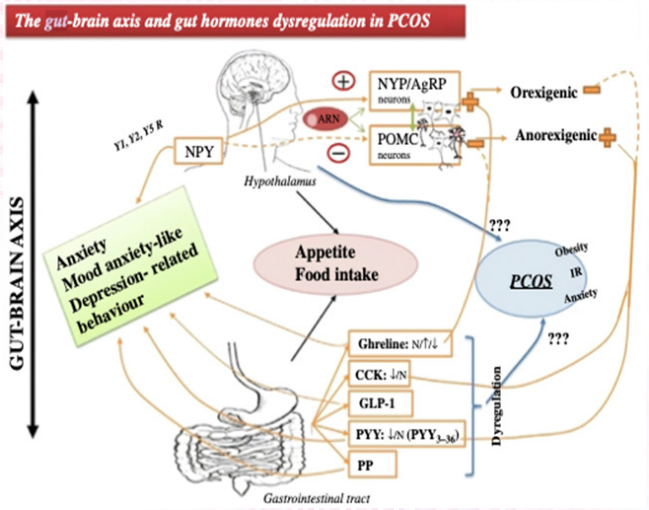

Figure 3. The gut -brain axis and gut hormones dys regulation in PCOS.[14]

Ghrelin,[4,14] cholecystokinin (CCK), and peptide YY(PYY) are important peptides for controlling eating and body weight.[4] It has been suggested that Glucagon-like peptides (GLP-1) and GLP-2, along with essential gut peptides, affect emotional-affective behavior. Evidence suggests ghrelin energizes behavior by stimulating the cholinergic-dopaminergic reward pathway. This link and hyperghrelinemia may provide a pharmacological treatment for binge eating.[4] On the contrary, studies showed that PYY encourages pleasurable behavior, as seen by the recognition that decreasing PYY intensifies depression-like features with no effect on anxiety. Moreover, PP, acting through Y4 receptors, may affect emotional-affective behavior. As for CCK, it has been linked to increased anxiety-like behavior in both humans and rodents. GLP-1 has reportedly been linked to heightened anxiety, while GLP-2 shown to lessen depression.[4] Lowering of CCK secretion may be linked to binge eating,[14] with decreased levels of CCK and ghrelin correlating to emotional and dietary issues in PCOS.[19] It has also been reported that appetite is impaired in women with PCOS, with alterations in hunger and gut hormones. A systematic review of 20 studies involving 894 women with and 574 women without PCOS reported lower ghrelin in PCOS (standardized mean difference -0.40; 95% confidence interval -0.73, -0.08), related to factors including insulin resistance.[26] In addition, research suggests women with PCOS tend to feel less full and hungrier after meals than women without PCOS, plus consuming more energy and hunger returning faster.[26] Other than ghrelin, all other gut hormones/peptides are usually associated with satiety.[27]

Eating Disorder in PCOS

Extensive research consistently shows a strong correlation between PCOS, disordered eating,[4,6,22,26] and the development of ED[24,26,28,29] in women. PCOS-related dietary changes, hunger, hyperinsulinemia, and obesity which present as disordered eating[14,30] can cause psychological problems such as anxiety, depression, cravings, overeating, and low self-esteem, and may be also caused by these psychological issues[14] (Figure 4).

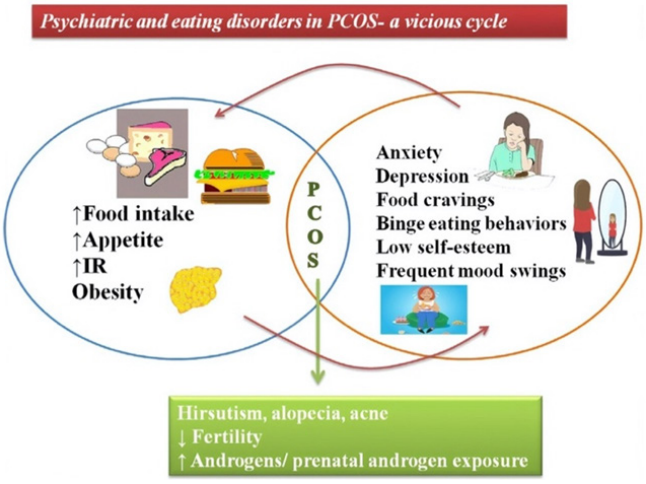

Figure 4. A vicious cycle of psychiatric and eating disorders in PCOS.[14]

A study of a meta-analysis established a high prevalence of depression and anxiety in PCOS sufferers, linked to elevated androgen and insulin levels.[12] In addition to these two essential elements, high stress raises cortisol, insulin, and androgen levels, resulting in depression and eating disorders due to their impact on mood and eating behavior.[13] These negative emotions increase the prevalence of disordered eating patterns.[31] Hyperandrogenism and hyperinsulinism commonly observed in PCOS women have greatly contributed to the development of eating disorders and abnormal eating habits in this population[18] (Figure 5).

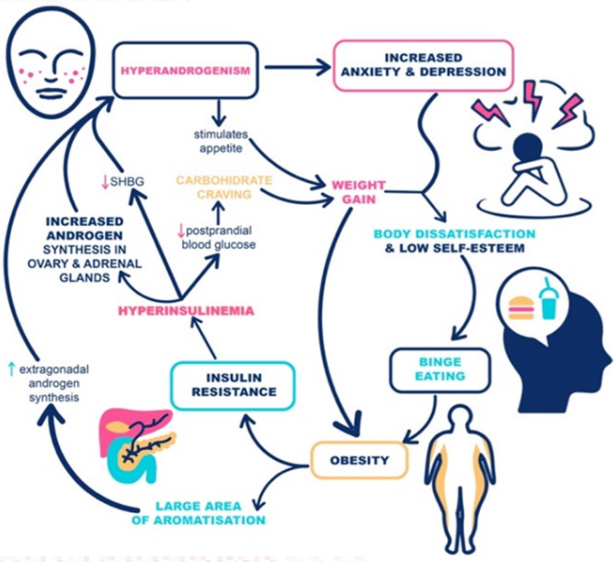

Figure 5 5Effects of hyper and rogenism and hyper insulinism on eating disorders in PCOS patients.[18]

According to research, high androgen levels may pose a heightened risk of BED[7,31] and BN.[28] These hormones may trigger bulimic behaviors through increased appetite[24,29] and decreased impulse control.[29] In actuality, compulsive overeating behaviors in PCOS patients with BED and BN are a way to cope with high stress.[13] PCOS patients who have BED often have elevated insulin levels, which can lower blood sugar thus stimulating hunger. Binge-eating impairs cognitive abilities and increases the likelihood of depression and anxiety.[31] Women with PCOS who have depression and anxiety are more likely to develop disordered eating than those without.[2] PCOS patients experience depression due to elevated androgen, insulin, and inflammation levels.[26] Research has revealed that high androgen levels are connected to more severe depression[12] and a fivefold increase in turbulent eating because of elevated anxiety.[2] Furthermore, a study suggests that hyperandrogenism may lead to a decrease in taste sensation in PCOS patients.[6] Elevated levels of testosterone, when combined with obesity, can often lead to intense hunger even when not hungry, potentially causing excessive eating and even eating-related issues.[29] PCOS, with its associated symptoms - acne,[8] hirsutism,[4,8] obesity, irregular periods, and infertility - has been linked to lower self-esteem[4,5] and body dissatisfaction, both of which can be causes of eating disorders.[5] Body image disturbance is a major factor in eating disorders, which can include irregular eating, restricted diets, and focus on body weight.[16] Excessive insulin and testosterone can spur an intense craving for carbs, signifying PCOS and BED[6] BED appears to be a factor in both obesity and disturbances in insulin regulation[7] Generally, fluctuating hormones during a menstrual cycle can influence food cravings and associated eating behavior[6] in PCOS women with ED.[30] Stress can cause a surge in appetite, resulting in overindulging and a higher Body Mass Index (BMI) in women with PCOS.[4] Nonetheless, it does not necessarily mean that all PCOS women are overweight/obese.[32] The frequency of overweight and obesity in patients frequently leads to disordered eating,[5] not just PCOS.[4] High BMI does not always imply an eating disorder, however, it may hint at low self-esteem[4] and physical distress that could alter eating habits.[4,5]

Research indicates genetic associations with serotonin, a neurotransmitter related to appetite, mood, and circadian rhythms, may be associated with disordered eating. Anorexia nervosa patients showed a drop in 5 HIAA levels, implying serotonin deficiency. This might result in a strict diet, as serotonin is created from tryptophan, an essential nutrient. Low serotonin and high testosterone in the brain may explain the aggressive behavior of those with eating disorders like BN. Research suggests testosterone modulates serotonin receptor activity, which impacts aggression, fear, and anxiety.[13]

BEDs are known to cause obesity which disrupts the appetite signaling system controlled by leptin and ghrelin hormones. Leptin, a hunger-reducing substance, had a major effect on hunger and fullness when administered in high doses to rodents, leading to decreased food consumption. However, the impact of leptin was short-lived, as the rodents displayed signs of resistance after two weeks, likely due to overproduction. Leptin affects reproductive processes through its effect on the hypothalamic-pituitary-ovarian (HPO) axis. Excess levels of leptin in overweight individuals may influence ovarian functioning.

Females afflicted with BN demonstrate a decreased sense of self-worth, a psychological factor, that may prompt them to be displeased with their physical form. Individuals who show intensified levels of anxiousness and depressive symptoms are prone to using disordered eating as an ineffectual way of dealing with their emotions. It is theorized that the adrenal hormones, notably cortisol and DHEA, increase in a depressive episode, and DHEA may have neuroprotective capabilities in the brain, restraining cortisol from damaging neurons and lessening the probability of developing psychopathology, and may even have a direct effect on depression. Imbalanced convictions about weight, body shape, and consumption are pivotal in the development and perpetuation of eating disorders.[12] To sum it all up, severely heightened eating disorders can drastically reduce the quality of life to a very alarming degree.[16]

COMPARISON BETWEEN ED TYPES & THEIR PREVALENCE

Table 1: shows the comparison between different types of eating disorders.

	BN	AN-BP	BED	OFSED
Age group	15-19 years and 20s	13-19 years and 20s	≥20 years	12-25 years
BMI status	>19.0 kg/m²	<17.5 kg/m²	>19.0 kg/m²	May not meet the BMI requirements
Compensatory behavior	Present	Present	May be present	Frequency of behaviors
Fear of weight gain	May be present	Present	May be present	
Distinctive features	Recurrent episodes of binges	Distortions in perceptions of weight/ size	-Associated with physical comorbidities -Binge eating episodes -Obese	Difficulty capturing its heterogenous nature

The most common eating disorder observed in women with PCOS is BED[4] No AN-BP diagnosis was observed in PCOS individuals.[4] A meta-analysis investigation revealed that there was no statistical relationship between PCOS and AN-BP. Both AN and PCOS cause patients to have abnormal eating habits and fluctuating weight, but there is a contrast between the two; PCOS is more common in women with normal or increased BMI, whereas AN is more common in underweight women. Furthermore, the same study discovered a link between PCOS and other types of eating disorders such as BN, BED, and any other ED.[25]

In a study conducted, researchers noted the prevalence of diagnosed EDs in PCOS patients. Results varied between diagnostic categories, with estimates for AN ranging from 0.0% to 1.3%, for BN ranging from 0.0% to 12.6%, for BED ranging from 4% to 15%, and one research reporting a frequency of 22.5% for EDNOS.[31]

DIFFERENCES BETWEEN DISORDERED EATING AND EATING DISORDER

Disordered eating is a condition that shares the same symptoms as an eating disorder but with a reduced frequency or degree of severity.[4,26,33] As explained earlier in this review, an eating disorder is a psychological disorder with abnormal eating behaviors.[26] Body image disturbances, anxiety towards specific food types, distorted perceptions, and low self-esteem based primarily on body weight have all been linked to disordered eating practices in PCOS patients.[4,16,26] Disordered eating appears to be used as a functional and emotional management technique by people who experience higher levels of anxiety and depressive symptoms.[2,5,11,12] In several studies examined, authors reported that when comparing PCOS patients to controls, disordered eating, but not eating disorders, were more common.[4,26] Different studies suggested different findings. One of them showed that the diagnosis of an eating disorder did not differ substantially between women with and without PCOS,[4] whilethe other collective study suggested an increased risk of both disordered eating and eating disorder in PCOS patients.[2] This is relevant because many women will report symptoms of disordered eating that are linked to discomfort and impairment, but they do not meet the diagnostic criteria for a particular ED diagnosis.[31] Furthermore, an earlier meta-analysis found that women with PCOS had a 3.05 odds ratio (OR) for disordered eating behavior when compared to those without.[4,31] To state another known relationship, after controlling for age and BMI in women with PCOS, a link was found with psychiatric comorbidities such that the presence of anxiety increases the likelihood of disordered eating by about fivefold,[2,34] and persistent weight gain over the follow-up period was also discovered.[11] It's interesting to note that the diagnosis of PCOS did not predict either the presence of disordered eating or the diagnosis of an eating disorder.[4] Irrespective of PCOS status, older ageand higher BMI increased the probability that women would engage in disordered eating behavior, whereas younger age and higher BMI increased the odds that women would be diagnosed with an eating disorder.[4] The cause of disordered eating in women with PCOS is unknown, however, it is believed to be influenced by several variables such as: higher BMI, weight, eating concerns,body dissatisfaction, anxiety, and higher diet restraint.[4,5]

The present evidence on either disordered eating or comprehensive ED diagnoses in women with PCOS suggests a significantly elevated risk.[34] However, in most of the articles, women with PCOS had more disordered eating behaviorsthanED.[4]

DIAGNOSTIC METHODS

PCOS

Rotterdam Criteria: According to the Rotterdam consensus, polycystic ovarian syndrome (PCOS) is diagnosed by the presence of two or three of the following criteria: 1) hyperandrogenism (clinical or biochemical) 2) oligo-anovulation 3) an ultrasound examination of polycystic ovaries.[3]

Eating Disorder

Eating disorder examination questionnaire (EDE-Q): The EDE-Q is a self-reported 28-item questionnaire and has the purpose of evaluating the range, regularity, and severity of behaviors used for the assessment of ED. The EDE-Q yields data loading on four subscales – Weight Concern, Shape Concern, Eating Concern, and Dietary Restraint as well as a Global score.[4,17]

The EDE-Q is the advised clinical gold standard screening instrument to evaluate the presence of essential cognitive and behavioral traits prevalent in both disordered eating and eating disorders.[4]

Diagnostic and statistical manual of mental disorders (DSM-5): It provides information on five different varieties of OSFED, including atypical AN, BN (of low frequency and/or limited length), BED (of low frequency and/or limited duration), purging disorder, and night eating syndrome.[30]

Eating attitudes test (EAT-26): The EAT-26 is perhaps the most extensively used standardized self-report assessment of eating disorder symptoms and concerns. It is a 26-item test that measures the concern about aberrant eating. A 6-point Likert scale was used by participants to rank each issue. Higher ratings indicating greater eating disorders.[8]

Demographics Questionnaire

A demographics questionnaire was utilized to gather background data, anthropometric measurements (such as height and weight), participant history, and eating disorder disclosure patterns.[17]

RACIAL DIFFERENCES

In one of the studies, the researchers correlated EDE-Q scores with behaviors of PCOS phenotypes. Similar comparisons were done across racial groups, defined as white vs. nonwhite, and then broken down into the following racial backgrounds in an exploratory analysis: "white", "black", "Asian", "Hispanic" or "mixed". There were notable disparities in EDE-Q scores between white and nonwhite women with PCOS. Nonwhite women exhibited higher global EDE-Q scores, thus indicating a greater burden of ED symptoms. Except for restraint, nonwhite women had greater subscale ratings than white women. Meanwhile, they found no difference in ED behavior rates between racial groups. In an exploratory analysis, they further classified race into white, black, Asian, Hispanic, and mixed racial groupings. Groups like black, Hispanic, and mixed women had higher EDE-Q global and subscale scores than white women; whereas, Asian women scored lower than white women. Cultural variations likely contribute to ED risk in many ways, such as through media, eating patterns, or beauty norms. Interconnected socioeconomic factors may also muddle the impact of race. Additional research is needed to determine whether and how race influences ED symptom risk in PCOS to develop culturally competent therapies in the future.[11]

FINDINGS

ED are more prevalent in women with PCOS. The frequency of eating disorders is substantially connected with amenorrhea ($p<0.001$) and oligomenorrhea ($p<0.001$),

according to a study conducted in Sweden on 11,503 women.[34] Researchers observed a high prevalence of BN, BED, and NES among the PCOS group using DSM-5 criteria, and the ED scores were noticeably higher for shape and weight worries.[34] They have also found a negative connection between EDE-Q global scores and PCOS screening test scores for health-related quality of life (HQoL).[34] Women with PCOS reported noticeably greater symptoms of anxiety and depression than controls and had low values in the measurement technique (HQoL) unlike the EDE-Q scores. Women with PCOS scored higher on the EDE-Q than a normative sample and reported that baseline measurements of hyperandrogenism, depressive symptoms, and BMI all predict disordered eating attitudes and behaviors.[11]

BED: A meta-analysis of four studies that included 868 participants revealed a statistically significant link between PCOS and a clinical diagnosis of binge eating disorder. (OR, 2.95; 95% CI, 1.61-5.42, I2=18.9%).[25]

BN: A meta-analysis of five trials with a total of 269,212 participants revealed a statistically significant link between PCOS and a clinical diagnosis of bulimia nervosa (OR, 1.37; 95% CI, 1.17-1.60; I2=0%). Utilizing multiple scales, three investigations with 458 individuals examined bulimia nervosa symptoms. Patients with PCOS performed better on these scales than patients without the condition. (standardized mean difference,1.25; 95% CI, 0.20-2.29; I2=95.1%).[25]

AN: A meta-analysis of three studies involving 268,417 participants found no statistically significant link between PCOS and anorexia nervosa. (OR, 0.92; 95% CI, 0.78-1.10; I2=0%).[25]

Any eating disorder:A meta-analysis of four research involving 7188 patients revealed a statistically significant link between PCOS and any clinically diagnosed eating disorder. (OR, 3.68; 95% CI, 1.38-9.81, I2=4 4. 5%).[25]

MANAGEMENT AND TREATMENT

For PCOS populations to experience the best physical results, early and accurate detection of binge eating symptoms is crucial,[30] also in order to rule out any mood or ED, women with PCOS would most likely need a full psychiatric evaluation.[19,36,37] The 2018 guidelines from the Androgen Excess-PCOS Association recognize the clinical importance of this problem and promote screening[2,5,35] for disordered eating in all PCOS-afflicted women.[30] Furthermore, it is critical to raise awareness[16] and educate women about disordered eating in PCOS, as many women with EDs get misdiagnosed and are unaware that their eating and weight-related beliefs and behaviors are abnormal and cause discomfort.[30]

The mainstay of PCOS treatment for women is a change in lifestyle.[2,4,38-40] It would be critical to monitor PCOS women's diet to aid with the symptoms caused by an excess of androgens and insulin resistance, as well as to reduce the risk of diabetes and heart disease later in life.[16,26,30,38] Several prospective studies have established that dieting predicts future bulimic symptomatology, and it is evident that when BN is present, treatment referral should focus on resolving the core BN symptoms rather than on weight loss.[15,34] Although these treatments have not yet been tested on PCOS women who also have eating disorders, it is anticipated that such an approach to weight management would be more effective than focusing solely on weight management because it would also address the main psychological factors underlying EDs.[4,31]

CONCLUSION

As a result of, clinicians should consider chronic psychological distress and eating disorders as significant factors when diagnosing PCOS. Furthermore, a comprehensive evaluation of eating behaviors and dietary patterns in females with PCOS is essential. Consequently, investigating the connection between PCOS and eating disorders holds great potential for improving the overall care and well-being of affected individuals.

ETHICAL DECLARATION

Referee Evaluation Process: Externally peer-reviewed.

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Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper and that they have approved the final version.

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